albumen we mark the omission of any statement relative to the forms of that substance which have an unusual reaction with heat and nitric acid; and also of the fact, very important in many cases, and carefully insisted upon by Beale, that when the quantity of albumen is small, either a very little or much nitric acid will dissolve it and prevent its detection. Dr. Flint's suggestion that the several solutions of Féhling's test for sugar be kept in separate bottles and combined as required, is a very good one. The gradual reduction of the copper and consequent vitiation of the mixed fluid is thus avoided. For estimating the amount of urea Davy's process is recommended, and Liebig's mentioned only by name. The author has found, however, that the results are not satisfactory if the American solution of chlorinated soda is used instead of the French. In the appendix there are directions for the analysis of urinary calculi most frequently met with, and tables to facilitate quantitative calculations. These tables are reprinted upon a single folded sheet at the end, which can be cut out and posted in a conspicuous place for easy reference. We regret that the author has followed Dr. Beale in substituting the embarrassing grains and fluidounces for grammes and cubic centimetres. Apparatus graduated according to the decimal system is readily obtained, and renders the whole proceeding much more simple. E. R.

ART. XXVI.—*The Dispensatory of the United States of America.* By GEORGE B. WOOD, M. D., President of the American Philosophical Society; President of the College of Physicians of Philadelphia; Emeritus Professor of the Theory and Practice of Medicine in the University of Pennsylvania, &c.; and FRANKLIN BACHE, M. D., Late Professor of Chemistry in Jefferson Medical College of Philadelphia; Late Vice-President of the College of Physicians of Philadelphia; Late President of the American Philosophical Society, &c. Thirteenth edition, carefully revised. 8vo. pp. 1810. Philadelphia: J. B. Lippincott & Co., 1870.

AN enlarged leaf, and about one hundred additional pages in the thirteenth issue of this great work, give us the measure of the rapid advance in pharmacological knowledge since the twelfth edition was published, only five years ago. Any analysis of the new matter would be superfluous. As the work has been done before, so is it continued now, and the profession will recognize with peculiar pleasure that the surviving author's skilful hand has lost none of its power. Everything that is of value concerning discoveries in pharmacy, recent uses of old remedies, and the introduction of new ones, will be found fully stated down to the latest possible date. The Dispensatory maintains its position as the most beneficent and generally useful medical book now in use.

E. R.

ART. XXVII.—*Ueber das Vorkommen Fibrinöser Entzündungs Producte in den Bronchien, und Lungen alveolen. Ueber Fibrinöse oder Pseudomembranöse Bronchitis und Pneumonie. Bronchitis Fibrinosa, Bronchitis Pseudo-membranacea, Pneumonia Fibrinosa.* Von Dr. LEBERT, Geheimerrath, und Professor in Breslau.

On the Product formed in the Bronchi and in the Air-cells of the Lungs in Cases of Inflammation of these Organs. On Fibrinous or Pseudo-membranous Bronchitis and Pneumonia, Bronchitis Fibrinosa, Bronchitis Pseudo-membranacea, Pneumonia Fibrinosa.

UNDER the above title, Professor Lebert has published in the *Deutsches Archiv für Klinische Medicin*, running through the numbers for 1869, a very complete, able, and instructive monograph in relation to a form of disease with which our physicians, as a body, would seem to be but slightly conversant, or, at least, to be little aware of the frequency of its occurrence in connection with ordinary forms of intra-thoracic inflammation, and the modifications it imparts when present to their symptoms, march, and termination. Believing that our readers will be gratified by having laid before them a general analysis of the views advanced by Professor L. in this admirable monograph, we have endeavoured to present one in such a form as shall do justice to the author, and, at the same time, by its conciseness, adapt it to the necessarily limited space appropriated to the bibliographical department of this Journal.

The fibrinous product met with so often in the inflammatory conditions of the bronchi and air-cells of the lungs, mark out, according to our author, the cases in which these conditions occur, a morbid affection, different entirely from, and unconnected with what has been denominated diphtheritis. It must be distinguished also from the results of those somewhat rare, or more properly very doubtful, cases, in which the diphtheritic inflammation, commencing in the throat and larynx, is supposed to extend thence downwards into the finest bronchial ramifications.

During childhood there occurs frequently, in attacks of ordinary bronchitis and of broncho-pneumonia, fibrinous formations in the bronchial ramifications, and yet without the presence of any very characteristic symptoms to indicate their presence, and without the occurrence of any fibrinous shreds in the sputa. In such cases the fibrinous formations are to be looked upon in the light of a mere complication, which may, however, in most cases, augment the suffocative character of catarrhal pneumonia, and also increase the danger of the attack.

There is a single case upon record, that of Hayn, in which the bronchial ramifications and pulmonary cells were, in a new-born infant, clogged with fibrinous deposits. This case proves the possibility of fibrinous bronchitis or fibro-pneumo-bronchitis occurring during intra-uterine life and the first days of infantile existence.

Professor L. describes the form of disease he considers as an acute, essentially fibrinous bronchitis, which, it is true, he admits, considered ontologically, exhibits no very sharply defined pathognomonic characteristics; nevertheless, a very little examination will show its essential character. It occurs twice as often in males as in females, more frequently also during childhood and youth than in after years. It very seldom occurs in the aged. This form of disease is characterized by severe, prolonged paroxysms of coughing, extreme difficulty of respiration, amounting, in some cases, apparently to impending suffocation, with often bloody sputa. The cough and difficulty of breathing are greatly relieved upon the discharge by the mouth of masses of fibrinous matter. This discharge usually occurs between the fourth and sixth day of the attack, in some cases not until the tenth or fourteenth day, or in other very rare cases again, not until the end of the third or fourth week. The discharge may take place only once, or be repeated several times a day. In the acute form of the disease the ejection of fibrinous matter may continue from seven to ten days, but its frequency diminishes usually from the second to the fourth day.

When first expectorated, the fibrinous masses are so intimately incorporated with mucus or blood, that they are very liable to be overlooked. They become much more apparent under water. They will then be seen ramifying in the form of tubes or cylinders, which divide and subdivide until, finally, they acquire a capillary fineness; often, at one end, there occurs a thickening, forming, as it were, a bulb. They are either hollow or solid, and consist for the most part of consolidated fibrinous matter, inclosing numerous white blood corpuscles (pus corpuscles) and a few epithelial cells.

Special auscultatory signs are seldom present in the form of disease under consideration. The following signs have, however, been adduced as diagnostic: A clear sound at a part of the chest where no distension is observable, and where the respiratory movement is very weak, scarcely audible, or when, over a small circumscribed space, the respiratory murmur is audible. Intrabronchial rattling, friction sound, a valve-like sound, as though a membranous partition sank and rose in inspiration and expiration.

The disease may occur in a mild or in a severe form. The first being attended with moderate and unfrequent paroxysms of coughing and difficulty of breathing; the second, on the contrary, is marked by frequent and intense paroxysms of almost impending suffocation. It is in this latter form that a fatal termination is the most to be feared. Professor L. tells us, as the result of his experience, that the deaths are in the proportion of four in seventeen cases. In only three was death produced by asphyxia. In fourteen cases it was consequent upon adynamic typhoid collapse.

The disease may run its course rapidly, while in other instances it may be somewhat prolonged. It may have a duration of from one week to one week and a half, or it may run on for three or four weeks or even longer. When death takes place, it ordinarily occurs between the sixth and twelfth days. The diffuse vesicular pneumonia, which is becoming to be more carefully distinguished from an original dissemination of catarrhal bronchitis, though, it is true, only a kind of intermediate morbid condition, resulting from a transition of one disease into the other, may occur, as was pointed out forty years ago by Lobstein of Strasburg, under the name of *croupal* bronchitis. A case in point is referred to by Morgagni. This theory of croupal, or more correctly, of fibrinous bronchitis or pneumonia is also sustained by Rokitansky.

We are to recollect that, as was pointed out some twenty-five years ago by Remak, that small fibrinous bronchial concretions are almost invariably met with in the proper sputa of ordinary pneumonia, and further, that even large fibrinous masses are occasionally coughed up in simple pneumonia the same as in the acute fibrinous form. In proof of this fact there has been put on record a series of cases in which a correct diagnosis has been clearly established: one by Morgagni, one by Oppolzer, one by Barth, and one by Professor L. In cases of ordinary pneumonia, fibrinous expectoration may occur in the course of the disease if protracted.

Not less interesting is another group of cases of acute pneumonia, in which, usually, there are formed small bronchial and pneumo-cellular fibrinous concretions. The disease occupies usually the entire bronchi of one lobe of a lung. Morgagni records a case of this kind; and a highly interesting series of similar cases is described by Nonat as having occurred during an epidemic of influenza, in patients especially who were convalescing. Many cases are related as having been treated at Schützenberger's clinic at Strasburg. This form of pneumonic disease is described by Professor L. in his work on *Pathological Anatomy*, where he gives a drawing also of the fibrinous product found in the lungs. Professor L. thinks it probable that it is to the presence of this fibrinous deposit in the lungs that the fatal result is generally due in those cases of pneumonia which eventuate in death.

The chronic form of idiopathic fibrinous bronchitis is met with more often in males than in females, in the proportion of nearly three to two. It occurs more often also during the earlier than during the later periods of life. It is seldom met with in persons over sixty years of age. They who are most liable to its attacks are such as, in popular phrase, "take cold easily," who are the subjects of chronic catarrh, or who are liable to frequent exposure to a cold and damp atmosphere. There would seem to exist, apparently, a predisposition to the disease in all the members of certain families.

The attack commences usually as one of simple catarrh, only assuming in its course those symptoms which stamp upon it its fibrinous character. The latter may, however, become apparent with great promptness. Often the expectoration of fibrinous matter is preceded by hæmoptysis. Severe paroxysms of coughing, and a sense of impending suffocation, may precede the expectoration of fibrinous matter, and be frequently repeated; at each repetition, however, abating gradually in violence. In prolonged cases the leading symptoms are such as to lead to a suspicion of the presence of tubercles in the lungs.

Chronic fibrinous bronchitis may be developed after repeated attacks of the disease in its acute or subacute form, occurring at very different, and sometimes prolonged intervals. During these attacks, before the fibrinous discharge takes place, the vital capacity of the lungs may become very much diminished, but, after the coughing-up of the excreted fibrinous matter, it is liable again to return to its normal condition. The physical signs of the chronic form of fibrinous bronchitis are the same as in the acute form.

The ordinary termination of the disease, even after long years of duration, is usually favourable. In twenty-seven cases, three, or eleven per cent., terminated fatally during a fit of strangulation.

It is to be kept in mind that the chronic form of fibrinous bronchitis may occur as a symptomatic affection during the course of some other disease. Seven well-marked cases of this are on record, in one of which it came on in a case of syphilis. Deep-seated ulceration of the trachea and at its place of bifurcation will cause bronchial contraction, dilatation ensuing, however, upon the expectoration of a membraniform substance. Deep-seated ulcers of the respiratory tubes Prof. L. has met with only as a result of syphilis. In one case the chronic fibrinous bronchitis occurred in conjunction with fatty degeneration of the heart with partly gangrenous and partly purulent pneumonia; in a third case it occurred in connection with empyema; and in four others with pulmonary tuberculosis. Chronic disease of the lungs, more than any other malady, would seem, therefore, to give a predisposition to the occurrence of chronic fibrinous bronchitis.

The pathogeny of abnormal fibrinous productions in the bronchi and pulmonary cells is of a far more extensive and common occurrence than is ordinarily supposed. Whenever inflammation becomes developed in these parts, as has been correctly stated, its product is inclined to assume a fibrinous character. In cases of common pneumonia it is in the nearest bronchial tubes that the fibrinous character is first developed. It is entirely devoid, however, of anything specific in its character.

The mechanism of the formation of these fibrinous bodies in inflammation of the air passages is, according to our author, as follows: premising that, according to Cohnheim, the fact being confirmed by the more recent experiments of Prof. L., the so-called pus-cells are, for the most part, free white blood corpuscles. With the escape of these *leucocytes*, there exudes, at the same time, a spontaneously coagulable albuminoid fluid. Its consolidation, apparently is the first to take place, being followed by the solidification of a fibrinous fluid, exuded simultaneously with it, which includes or envelops the free white cells and a portion of the adjacent epithelium. The fibrinous formation thus fashioned is perfected by a quick repetition of the same processes. In general, the exudated fibrogenetic fluid appears to be very intimately connected with the formation of the white blood cells. Prof. L. never saw an instance in which red blood corpuscles were found to be imbedded in these fibrinous formations. A proof of this is furnished by the sputa proper to pneumonia; separate its different components, and it will be found that red blood cells exist only in the more slimy portion, which is abundant in mucus, and secreted solely by the glandulæ of the bronchial mucous membrane. In this viscous, tenacious fluid, more or less coloured by the presence of red blood corpuscles, will be found the small, tubular ramifications mechanically entangled together, and imbedded in a granular, filamentous basis, in company with many round, enucleated cells and white blood corpuscles, which, upon the application of acetic acid, are rendered more apparent, but without any admixture of red blood cells.

That not only diffused cellular pneumonia, but, also, the catarrhal broncho-pneumonia of early life has a very decided disposition to assume the fibrinous form is proved by the fact of the relative frequency with which we encounter, at the bedside, the fibrinous formation as an anatomical complication in the latter disease during childhood. Nonat's cases, which occurred during the prevalence of epidemic catarrh at Paris, in 1837, exhibited, as it were, a kind of transition from one to the other, of the two leading forms of inter-bronchial fibrinous deposit.

Fibrinous deposits may be absent entirely in cases of ordinary bronchitis, even when occurring in young persons; even the most severe cases of pneumonia may exhibit a relatively small fibrogenetic tendency, without our being able to discover for this any immediate cause. Prof. L. believes that, in some cases, there may be a particular predisposition to fibrinous deposits, present in some persons, absent in others. He has known several sisters of a family, all of whom were simultaneously attacked with fibrinous bronchitis. Some peculiarity in the local inflammatory process may, it is not absurd to suppose, also exert a certain amount of influence, so that a proportionately greater amount of fibrogenetic fluid is made to accumulate in the vessels at the seat of the inflammation than is the case in inflammatory processes occurring in other individuals; as a consequence of this state of things, it is reasonable to believe, that a much greater amount of fibrinous matter will be poured out into the bronchi or pulmonary cells—the excretion of fibrinous matter being an item in the ordinary course of the disease.

This state of things—the overfilling of the inflamed vessels with fibrinous matter—may not be remedied by the exudation of the fibrogenetic matter, and then the true character and location of the attack is first developed; from which epoch its course may be prolonged for days, or even weeks, and, assuming a chronic form, from one or many years, with varying length of intervals between its accessions. Even after bronchial inflammation has been arrested, it may be reinduced readily, with its fibrinous character. After the lapse of years, however, the local predisposition may finally cease.

Fibrinous bronchitis has, by some writers, been pronounced a severe form of croup, resulting from the spreading downwards, into the trachea, bronchi, and pulmonary cells, of the croupal inflammation, commencing at the fauces. This view of the form of the disease in question is not so common now as formerly. It is true that in cases of pneumonia in the smaller bronchial ramifications fibrinous products may occur, and from thence spread throughout all the bronchi, even to the upper bronchial bifurcation. This has been proven by recent observations recorded by Lücken and Zieful. In cases where the ordinary expectoration of small coagula, as are seen in pneumonia, and which are not replaced by fine capillary filaments, upon an anatomical examination of the hepatized lung there will be found in the proper pulmonary cells only a moderate accumulation of cysts, as it were, completely glued together, and presenting no trace of an intermediate fibrinous substance, resembling the fibrinous concretions of the bronchial ramifications. We observe, further, in cases of true fibrinous bronchitis, as in pneumonia, large concretions discharged by coughing, terminating very often in very fine, almost capillary ramifications, the finest having a diameter of 1–2 mm.; this, it is true, is often absent. It, is, therefore, very probable that the fibrinous formations in the bronchi are of very different diameters.

It is very evident that, amid all the varieties exhibited by fibrinous inflammations of the respiratory organs, there present themselves two very distinctly marked forms of true fibrinous bronchitis, having their anatomical bases in the more or less disposition to, the extent of surface occupied by, or the more or less persistence of, the fibrogenetic morbid action. The amount of fibrinous deposit, the nature and extent of its divisions and bifurcations; the adhesiveness of the entire mass, are all disproportionate to the excretory power of the bronchi, and therefore decrease the vital respiratory capacity of the lungs. Hence the great difficulty of breathing, the necessity of increased and more forcible efforts to relieve the bronchi of their oppressive load, by severe paroxysms of coughing, the frequent rupture of small bloodvessels, and the impending sense and danger of suffocation. Very often the fibrogenetic action diminishes or ceases, subsequent to frequent copious and intense paroxysms of expectoration, and a condition of general convalescence sets in.

Fibrinous bronchitis, as above described, exhibits as a secondary disease of the respiratory organs, the utmost multifariousness, in different cases, as to its causes and the extent of bronchial fibrogenesis. It is a well-established fact that fibrinous bronchitis may occur during all and every attack of inflammation of the respiratory organs, as was pointed out many years since, by Prof. L. Now a form of disease, like the one under discussion, having so different and diversified pathological bases, must necessarily become, every day, in the view of all reflecting physicians, stripped of its character, as an essential malady. Inflammation itself is, in fact, experiencing, in this respect, since the commencement of the present century, the same lot which has befallen the former supposed essential character of fever.

D. F. C.